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Reports on Selected Racial/Ethnic Groups

Contents

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Contents

Foreword

The purpose of the *CDC Surveillance Summaries* is to make available the most current information on conditions of public health interest for which CDC has major responsibility. The reports in this publication complement data provided in the *Morbidity and Mortality Weekly Report (MMWR)* and other CDC publications.

For information on the history of CDC surveillance activities, data sources, and surveillance systems, including the dates of the most recently published reports, refer to *CDC Surveillance Summaries* 1988;37(SS-2), dated June 1988.

Distribution of AIDS Cases, by Racial/Ethnic Group and Exposure Category, United States, June 1, 1981-July 4, 1988

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INTRODUCTION

Of the 66,464 cases of acquired immunodeficiency syndrome (AIDS) reported to CDC in the period June 1, 1981-July 4, 1988, most (60%) occurred among non-Hispanic whites; however, blacks and Hispanics accounted for 70% of the cases in heterosexual men, 70% of those in women, and 75% of those in children. To study the association between AIDS and racial/ethnic groups, the AIDS Program, Center for Infectious Diseases, analyzed the presumed means by which each patient became infected with human immunodeficiency virus (HIV) (i.e., his or her exposure category).

METHODS

CDC receives AIDS case reports from the health departments of all 50 states, the District of Columbia, and U.S. territories. This analysis included only cases meeting the CDC case definition (*1*). Thirty-six cases in U.S. territories other than Puerto Rico were excluded because of small numbers. The distribution of cases by racial/ethnic group and exposure category were analyzed. The racial/ethnic groups consisted of Hispanics and the following groups of non-Hispanics: whites, blacks, Asians and Pacific Islanders (Asians/PIs), and American Indians and Alaskan Natives (American Indians/ANs). AIDS patients residing in Puerto Rico (97% Hispanic) were studied separately from other Hispanics. Except for the combination of intravenous-drug abuse (IVDA) in homosexual/bisexual men, the exposure categories were hierarchically ordered so that persons with more than one possible means of acquiring HIV infection were classified only in the category listed first.

RESULTS

Analysis of the AIDS cases reported in the United States was limited to the 65,133 (99.8%) cases in which racial/ethnic group was specified. U.S. AIDS patients were disproportionately black (26%) and Hispanic (13%), compared with the proportions of blacks (12%) and Hispanics (6%) in the U.S. population (*2*).

The proportion of AIDS cases in which the mode of HIV exposure was homosexual activity among men was lower for U.S. black, Hispanic, and American Indian/AN AIDS patients and for AIDS patients in Puerto Rico than for U.S. white or Asian/PI AIDS

patients (Table 1). Heterosexual men and women constituted the majority of AIDS cases in U.S. blacks and residents of Puerto Rico.

Among men with AIDS, the proportion who were heterosexual intravenous-drug abusers or whose female sex partners were intravenous-drug abusers was 34% for U.S. black men, 35% for U.S. Hispanic men, and 52% for men in Puerto Rico, compared with 5%, 2%, and 10% for U.S. white, Asian/PI, and American Indian/AN men, respectively (Table 2). Among women with AIDS, the proportion who were intravenous-drug abusers or whose male sex partners were intravenous-drug abusers was 74% and 80% for U.S. black and Hispanic women, respectively, and 83% for women in Puerto Rico, compared with 52%, 31%, and 50% for U.S. white, Asian/PI, and American Indian/AN women, respectively (Table 3). Among children with AIDS, the proportion whose mothers or mothers' sex partners were intravenous-drug abusers was 62% and 72%, respectively, for U.S. black and Hispanic children and 81% for children in Puerto Rico, compared with 31%, 25%, and 50% for U.S. white, Asian/PI, and American Indian/AN children, respectively (Table 4). Overall, the proportion of AIDS cases associated with IVDA by heterosexuals (including sex partners and children of drug abusers) was 42% for U.S. blacks, 40% for U.S. Hispanics, and 58% for residents of Puerto Rico, compared with 7% for U.S. whites, 5% for U.S. Asians/PIs, and 19% for American Indians/ANs.

Of all U.S. AIDS cases associated with IVDA by heterosexuals, 54% occurred among blacks and 26% among Hispanics. Similarly large proportions of blacks and Hispanics were found among men, women, and children with IVDA-associated AIDS (Tables 5-7).

DISCUSSION

The data presented here support the findings of earlier analyses (*3-8*) that AIDS patients are disproportionately black and Hispanic and that the proportion of IVDA-associated AIDS cases is substantially greater in U.S. blacks and Hispanics than in U.S. whites. The disproportionate numbers of blacks and Hispanics treated for heroin abuse (*9*) suggest that they may have a higher prevalence of IVDA than whites. Black and Hispanic communities in the United States and Puerto Rico should be especially targeted for measures to prevent HIV transmission by treating drug abusers and by counseling drug abusers and their sex partners on the risk of HIV infection. Recommendations for preventing HIV transmission to intravenous-drug abusers, their sex partners, and their children have been published (*10-14*).

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TABLE 1. Distribution of AIDS patients, by category and racial

U.S.

Category	White		Black		Hispanic	
	No.	(%)	No.	(%)	No.	(%)
Homosexual or bisexual men	33,204	(85.3)	7,532	(43.7)	4,431	(52.0)
Heterosexual men*	3,963	(10.2)	6,429	(37.3)	3,062	(36.0)
Women	1,506	(3.9)	2,729	(15.8)	821	(9.6)
Children (<13 years)	247	(0.6)	557	(3.2)	202	(2.4)
Total	**38,920**	**(100.0)**	**17,247**	**(100.0)**	**8,516**	**(100.0)**

*Regardless of means of exposure to HIV.

racial/ethnic group, United States and Puerto Rico, July 4, 1988

U.S. residents								
ic		Asian/ Pacific Islander		Am. Indian/ Alaskan Native		U.S. Total		Puerto Rican residents
(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
(52.0)	297	(76.9)	40	(62.5)	45,504	(69.9)	397	(34.9)
(36.0)	56	(14.5)	10	(15.6)	13,520	(20.8)	535	(47.0)
(9.6)	29	(7.5)	10	(15.6)	5,095	(7.8)	169	(14.8)
(2.4)	4	(1.0)	4	(6.2)	1,014	(1.6)	37	(3.2)
100.0)	386	(100.0)	64	(100.0)	65,133	(100.0)	1,138	(100.0)

TABLE 2. Distribution of AIDS cases in men, by exposure category
July 4, 1988

						U.S.
	White		Black		Hispanic	
Exposure category*	No.	(%)	No.	(%)	No.	(%)
Homosexual or bisexual men without IVDA†	30,254	(81.4)	6,341	(45.4)	3,907	(52.1
Heterosexual men with IVDA	1,799	(4.8)	4,666	(33.4)	2,596	(34.6
Homosexual or bisexual men with IVDA	2,950	(7.9)	1,191	(8.5)	524	(7.0
Men with coagulation disorder (e.g., hemophilia)	526	(1.4)	35	(0.2)	39	(0.5
Heterosexual men whose sex partners had IVDA	89	(0.2)	142	(1.0)	29	(0.4
Heterosexual men born in certain countries§	4	(0.0)	807	(5.8)	4	(0.0
Heterosexual men whose sex partners were born in certain countries§	14	(0.0)	11	(0.1)	1	(0.0
Heterosexual men whose sex partners were HIV+ transfusion recipients	5	(0.0)	5	(0.0)	0	(0.0
Heterosexual men whose sex partners had HIV infection but undetermined means of acquiring it	31	(0.1)	21	(0.2)	5	(0.1
Heterosexual men whose sex partners had unspecified type of high risk¶	6	(0.0)	4	(0.0)	1	(0.0
Transfusion recipients	815	(2.2)	145	(1.0)	65	(0.9
Men with undetermined means of acquiring HIV infection**	674	(1.8)	593	(4.2)	322	(4.3
Total	**37,167**	**(100.0)**	**13,961**	**(100.0)**	**7,493**	**(100.0**

*Exposure categories are hierarchically ordered; persons with multiple me listed first, except for homosexual/bisexual men with IVDA.
†IVDA = history of intravenous drug abuse.
§Countries (e.g., Haiti, central African countries) in which heterosexual trans of transmission have not been fully defined.
¶Under investigation.
**Includes patients on whom risk information is incomplete (because of dea cases are still under investigation, and patients for whom (after complete

egory and racial/ethnic group, United States and Puerto Rico,

U.S. residents								
anic	Asian/ Pacific Islander		Am. Indian/ Alaskan Native		U.S. Total		Puerto Rican residents	
(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
(52.1)	290	(82.2)	31	(62.0)	40,823	(69.2)	240	(25.8)
(34.6)	7	(2.0)	5	(10.0)	9,073	(15.4)	484	(51.9)
(7.0)	7	(2.0)	9	(18.0)	4,681	(7.9)	157	(16.8)
(0.5)	6	(1.7)	3	(6.0)	609	(1.0)	3	(0.3)
(0.4)	1	(0.3)	0	(0.0)	261	(0.4)	1	(0.1)
(0.0)	2	(0.6)	0	(0.0)	817	(1.4)	0	(0.0)
(0.0)	0	(0.0)	0	(0.0)	26	(0.0)	1	(0.1)
(0.0)	0	(0.0)	0	(0.0)	10	(0.0)	0	(0.0)
(0.1)	0	(0.0)	0	(0.0)	57	(0.1)	0	(0.0)
(0.0)	0	(0.0)	0	(0.0)	11	(0.0)	0	(0.0)
(0.9)	20	(5.7)	0	(0.0)	1,045	(1.8)	11	(1.2)
(4.3)	20	(5.7)	2	(4.0)	1,611	(2.7)	35	(3.8)
(100.0)	**353**	**(100.0)**	**50**	**(100.0)**	**59,024**	(100.0)	932	**(100.0)**

ie means of acquiring HIV infection are tabulated only in the category

l transmission is believed to play a major role, although precise means

of death, refusal to be interviewed, or loss to follow-up), patients whose
plete investigation) no specific risk factor was identified.

**TABLE 3. Distribution of AIDS cases in women, by exposure categ
July 4, 1988**

	White		Black		Hispanic	
Exposure category*	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
Women with IVDA†	614	(40.8)	1,574	(57.7)	425	(51.8)
Women with coagulation disorder (e.g., hemophilia)	13	(0.9)	4	(0.2)	0	(0.0)
Women whose male sex partners had IVDA	165	(11.0)	450	(16.5)	233	(28.4)
Women whose male sex partners were bisexual	101	(6.7)	60	(2.2)	25	(3.0)
Women whose male sex partners had hemophilia	21	(1.4)	1	(0.0)	1	(0.1)
Women born in certain countries§	0	(0.0)	237	(8.7)	3	(0.4)
Women whose male sex partners were born in certain countries§	0	(0.0)	19	(0.7)	1	(0.1)
Women whose male sex partners were HIV+ transfusion recipients	20	(1.3)	3	(0.1)	2	(0.2)
Women whose male sex partners had HIV infection but undetermined means of acquiring it	42	(2.8)	53	(1.9)	15	(1.8)
Women whose male sex partners had unspecified type of high risk¶	2	(0.1)	5	(0.2)	4	(0.5)
Transfusion recipients	406	(27.0)	111	(4.1)	41	(5.0)
Women with undetermined means of acquiring HIV infection**	122	(8.1)	212	(7.8)	71	(8.6)
Total	**1,506**	**(100.0)**	**2,729**	**(100.0)**	**821**	**(100.0)**

*Exposure categories are hierarchically ordered; persons with multiple r
listed first.
†IVDA = history of intravenous drug abuse.
§Countries (e.g., Haiti, central African countries) in which heterosexual tra
of transmission have not been fully defined.
¶Under investigation.
**Includes patients on whom risk information is incomplete (because of d
cases are still under investigation, and patients for whom (after complet

category and racial/ethnic group, United States and Puerto Rico,

U.S. residents								
nic	Asian/ Pacific Islander		Am. Indian/ Alaskan Native		U.S. Total		Puerto Rican residents	
(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
(51.8)	7	(24.1)	5	(50.0)	2,625	(51.5)	102	(60.4)
(0.0)	0	(0.0)	0	(0.0)	17	(0.3)	1	(0.6)
(28.4)	2	(6.9)	0	(0.0)	850	(16.7)	38	(22.5)
(3.0)	2	(6.9)	0	(0.0)	188	(3.7)	1	(0.6)
(0.1)	1	(3.4)	0	(0.0)	24	(0.5)	0	(0.0)
(0.4)	0	(0.0)	0	(0.0)	240	(4.7)	0	(0.0)
(0.1)	0	(0.0)	0	(0.0)	20	(0.4)	0	(0.0)
(0.2)	0	(0.0)	0	(0.0)	25	(0.5)	0	(0.0)
(1.8)	2	(6.9)	2	(20.0)	114	(2.2)	1	(0.6)
(0.5)	0	(0.0)	0	(0.0)	11	(0.2)	0	(0.0)
(5.0)	11	(37.9)	2	(20.0)	571	(11.2)	13	(7.7)
(8.6)	4	(13.8)	1	(10.0)	410	(8.0)	11	(6.5)
(100.0)	**29**	**(100.0)**	**10**	**(100.0)**	**5,095**	**(100.0)**	**169**	**(100.0)**

tiple means of acquiring HIV infection are tabulated only in the category

ual transmission is believed to play a major role, although precise means

e of death, refusal to be interviewed, or loss to follow-up), patients whose mplete investigation) no specific risk factor was identified.

TABLE 4. Distribution of AIDS cases in children (<13 years), by exp
Puerto Rico, July 4, 1988

	U.S					
	White		Black		Hispanic	
Exposure category*	No.	(%)	No.	(%)	No.	(%)
Children with coagulation disorder (e.g., hemophilia)	44	(17.8)	6	(1.1)	8	(4.0)
Children whose mothers had IVDA†	54	(21.9)	265	(47.6)	107	(53.0)
Children whose mothers' sex partners had IVDA	22	(8.9)	79	(14.2)	39	(19.3)
Children whose mothers' sex partners were bisexual men	10	(4.0)	9	(1.6)	2	(1.0)
Children whose mothers' sex partners had hemophilia	4	(1.6)	1	(0.2)	1	(0.5)
Children whose mothers were born in certain countries§	2	(0.8)	89	(16.0)	1	(0.5)
Children whose mothers' sex partners were born in certain countries§	0	(0.0)	2	(0.4)	0	(0.0)
Children whose mothers' sex partners had HIV infection but undetermined means of acquiring it	7	(2.8)	12	(2.2)	7	(3.5)
Children whose mothers were HIV+ transfusion recipients	7	(2.8)	9	(1.6)	4	(2.0)
Children whose mothers had HIV infection but undetermined means of acquiring it	11	(4.4)	30	(5.4)	5	(2.5)
Transfusion recipients	77	(31.2)	31	(5.6)	23	(11.4)
Children with undetermined means of acquiring HIV infection¶	9	(3.6)	24	(4.3)	5	(2.5)
Total	**247**	**(100.0)**	**557**	**(100.0)**	**202**	**(100.0)**

*Exposure categories are hierarchically ordered; persons with multiple m
listed first.
†IVDA = history of intravenous drug abuse.
§Countries (e.g., Haiti, central African countries) in which heterosexual tran
of transmission have not been fully defined.
¶Includes patients on whom risk information is incomplete (because of pare
cases are still under investigation, and patients for whom (after complete

y exposure category and racial/ethnic group, United States and

	U.S. residents							
	Asian/ Pacific Islander		Am. Indian/ Alaskan Native		U.S. Total		Puerto Rican residents	
%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
(4.0)	2	(50.0)	0	(0.0)	60	(5.9)	1	(2.7)
(3.0)	1	(25.0)	2	(50.0)	429	(42.3)	17	(45.9)
9.3)	0	(0.0)	0	(0.0)	140	(13.8)	13	(35.1)
(1.0)	0	(0.0)	0	(0.0)	21	(2.1)	0	(0.0)
(0.5)	0	(0.0)	0	(0.0)	6	(0.6)	0	(0.0)
(0.5)	0	(0.0)	0	(0.0)	92	(9.1)	0	(0.0)
(0.0)	0	(0.0)	0	(0.0)	2	(0.2)	0	(0.0)
(3.5)	1	(25.0)	0	(0.0)	27	(2.7)	0	(0.0)
(2.0)	0	(0.0)	0	(0.0)	20	(2.0)	0	(0.0)
(2.5)	0	(0.0)	1	(25.0)	47	(4.6)	1	(2.7)
11.4)	0	(0.0)	1	(25.0)	132	(13.0)	4	(10.8)
(2.5)	0	(0.0)	0	(0.0)	38	(3.8)	1	(2.7)
00.0)	4	(100.0)	4	(100.0)	1,014	(100.0)	37	(100.0)

ple means of acquiring HIV infection are tabulated only in the category

al transmission is believed to play a major role, although precise means

of parents' refusal to be interviewed or loss to follow-up), patients whose
nplete investigation) no specific risk factor was identified.

TABLE 5. Distribution of AIDS cases in men, by exposure category a territories), July 4, 1988

Exposure category*	White		Black		Hispanic	
	No.	(%)	No.	(%)	No.	(%
Homosexual or bisexual men without IVDA†	30,254	(74.1)	6,341	(15.5)	3,907	(
Heterosexual men with IVDA	1,799	(19.8)	4,666	(51.4)	2,596	(2
Homosexual or bisexual men with IVDA	2,950	(63.0)	1,191	(25.4)	524	(1
Men with coagulation disorder (e.g., hemophilia)	526	(86.4)	35	(5.8)	39	(
Heterosexual men whose sex partners had IVDA	89	(34.1)	142	(54.4)	29	(1
Heterosexual men born in certain countries§	4	(0.5)	807	(98.8)	4	(
Heterosexual men whose sex partners were born in certain countries§	14	(53.8)	11	(42.3)	1	(
Heterosexual men whose sex partners were HIV+ transfusion recipients	5	(50.0)	5	(50.0)	0	(
Heterosexual men whose sex partners had HIV infection but undetermined means of acquiring it	31	(51.4)	21	(36.8)	5	(
Heterosexual men whose sex partners had unspecified type of high risk¶	6	(54.6)	4	(36.4)	1	(
Transfusion recipients	815	(78.0)	145	(13.9)	65	(
Men with undetermined means of acquiring HIV infection**	674	(41.8)	593	(36.8)	322	(2
Total	**37,167**	**(63.0)**	**13,961**	**(23.6)**	**7,493**	**(1**
Corresponding U.S. population in thousands††	71,495	(81.6)	9,128	(10.4)	5,199	(

*Exposure categories are hierarchically ordered; persons with multiple mean listed first.
†IVDA = history of intravenous drug abuse.
§Countries (e.g., Haiti, central African countries) in which heterosexual transm of transmission have not been fully defined.
¶Under investigation.
**Includes patients on whom risk information is incomplete (because of death, cases are still under investigation, and patients for whom (after complete inv
††1980 U.S. census data on males ≥13 years; data distinguishing Asians and were unavailable.

U.S. residents						
panic	Asian/ Pacific Islander		Am. Indian/ Alaskan Native		U.S. Total	
(%)	No.	(%)	No.	(%)	No.	(%)
(9.6)	290	(0.7)	31	(0.1)	40,823	(100.0)
(28.6)	7	(0.1)	5	(0.1)	9,073	(100.0)
(11.2)	7	(0.2)	9	(0.2)	4,681	(100.0)
(6.4)	6	(1.0)	3	(0.5)	609	(100.0)
(11.1)	1	(0.4)	0	(0.0)	261	(100.0)
(0.5)	2	(0.2)	0	(0.0)	817	(100.0)
(3.8)	0	(0.0)	0	(0.0)	26	(100.0)
(0.0)	0	(0.0)	0	(0.0)	10	(100.0)
(8.8)	0	(0.0)	0	(0.0)	57	(100.0)
(9.1)	0	(0.0)	0	(0.0)	11	(100.0)
(6.2)	20	(1.9)	0	(0.0)	1,045	(100.0)
(20.0)	20	(1.2)	2	(0.1)	1,611	(100.0)
(12.7)	**353**	**(0.6)**	**50**	**(0.1)**	**59,024**	**(100.0)**
(5.9)	Other††		1,803	(2.1)	87,625	(100.0)

means of acquiring HIV infection are tabulated only in the category

ansmission is believed to play a major role, although precise means

death, refusal to be interviewed, or loss to follow-up), patients whose
te investigation) no specific risk factor was identified.
s and Pacific Islanders from American Indians and Alaskan Natives

TABLE 6. Distribution of AIDS cases in women, by exposure categ territories), July 4, 1988

Exposure category*	White		Black		Hisp
	No.	(%)	No.	(%)	No.
Women with IVDA†	614	(23.4)	1,574	(60.0)	425
Women with coagulation disorder (e.g., hemophilia)	13	(76.5)	4	(23.5)	0
Women whose male sex partners had IVDA	165	(19.4)	450	(52.9)	233
Women whose male sex partners were bisexual	101	(53.7)	60	(31.9)	25
Women whose male sex partners had hemophilia	21	(87.5)	1	(4.2)	1
Women born in certain countries§	0	(0.0)	237	(98.8)	3
Women whose male sex partners were born in certain countries§	0	(0.0)	19	(95.0)	1
Women whose male sex partners were HIV+ transfusion recipients	20	(80.0)	3	(12.0)	2
Women whose male sex partners had HIV infection but undetermined means of acquiring it	42	(36.8)	53	(46.5)	15
Women whose male sex partners had unspecified type of high risk¶	2	(18.2)	5	(45.4)	4
Transfusion recipients	406	(71.1)	111	(19.4)	41
Women with undetermined means of acquiring HIV infection**	122	(29.8)	212	(51.7)	71
Total	**1,506**	**(29.6)**	**2,729**	**(53.6)**	**821**
Corresponding U.S. population in thousands††	77,185	(81.2)	10,614	(11.2)	5,322

*Exposure categories are hierarchically ordered; persons with multiple m listed first.
†IVDA = history of intravenous drug abuse.
§Countries (e.g., Haiti, central African countries) in which heterosexual tra of transmission have not been fully defined.
¶Under investigation.
**Includes patients on whom risk information is incomplete (because of de cases are still under investigation, and patients for whom (after complete
††1980 U.S. census data on females ≥13 years; data distinguishing Asians were unavailable.

ategory and racial/ethnic group, United States (excluding U.S.

U.S. residents							
Hispanic		Asian/ Pacific Islander		Am. Indian/ Alaskan Native		Total	
o.	(%)	No.	(%)	No.	(%)	No.	(%)
425	(16.2)	7	(0.3)	5	(0.2)	2,625	(100.0)
0	(0.0)	0	(0.0)	0	(0.0)	17	(100.0)
233	(27.4)	2	(0.2)	0	(0.0)	850	(100.0)
25	(13.3)	2	(1.1)	0	(0.0)	188	(100.0)
1	(4.2)	1	(4.2)	0	(0.0)	24	(100.0)
3	(1.2)	0	(0.0)	0	(0.0)	240	(100.0)
1	(5.0)	0	(0.0)	0	(0.0)	20	(100.0)
2	(8.0)	0	(0.0)	0	(0.0)	25	(100.0)
15	(13.2)	2	(1.8)	2	(1.8)	114	(100.0)
4	(36.4)	0	(0.0)	0	(0.0)	11	(100.0)
41	(7.2)	11	(1.9)	2	(0.4)	571	(100.0)
71	(17.3)	4	(1.0)	1	(0.2)	410	(100.0)
821	(16.1)	29	(0.6)	10	(0.2)	5,095	(100.0)
322	(5.6)	Other††		1,935	(2.0)	95,056	(100.0)

iple means of acquiring HIV infection are tabulated only in the category

al transmission is believed to play a major role, although precise means

e of death, refusal to be interviewed, or loss to follow-up), patients whose
mplete investigation) no specific risk factor was identified.
Asians and Pacific Islanders from American Indians and Alaskan Natives

**TABLE 7. Distribution of AIDS cases in children (<13 years), by expo
(excluding U.S. territories), July 4, 1988**

Exposure category*	White		Black		Hispanic	
	No.	(%)	No.	(%)	No.	(
Children with coagulation disorder (e.g., hemophilia)	44	(73.3)	6	(10.0)	8	(1
Children whose mothers had IVDA†	54	(12.6)	265	(61.8)	107	(2
Children whose mothers' sex partners had IVDA	22	(15.7)	79	(56.4)	39	(2
Children whose mothers' sex partners were bisexual men	10	(47.6)	9	(42.9)	2	(
Children whose mothers' sex partners had hemophilia	4	(66.7)	1	(16.7)	1	(1
Children whose mothers were born in certain countries§	2	(2.2)	89	(96.7)	1	(
Children whose mothers' sex partners were born in certain countries§	0	(0.0)	2	(100.0)	0	(
Children whose mothers' sex partners had HIV infection but undetermined means of acquiring it	7	(25.9)	12	(44.4)	7	(2
Children whose mothers were HIV+ transfusion recipients	7	(35.0)	9	(45.0)	4	(2
Children whose mothers had HIV infection but undetermined means of acquiring it	11	(23.4)	30	(63.8)	5	(1
Transfusion recipients	77	(58.3)	31	(23.5)	23	(1
Children with undetermined means of acquiring HIV infection¶	9	(23.7)	24	(63.2)	5	(1
Total	**247**	**(24.4)**	**557**	**(54.9)**	**202**	**(1**
Corresponding U.S. population in thousands**	32,226	(73.5)	6,399	(14.6)	4,088	(

*Exposure categories are hierarchically ordered; persons with multiple means listed first.
†IVDA = history of intravenous drug abuse.
§Countries (e.g., Haiti, central African countries) in which heterosexual transmis of transmission have not been fully defined.
¶Includes patients on whom risk information is incomplete (because of parents' cases are still under investigation, and patients for whom (after complete inve
**1980 U.S. census data on children <13 years; data distinguishing Asians and were unavailable.

xposure category and racial/ethnic group, United States

U.S. residents						
panic	Asian/ Pacific Islander		Am. Indian/ Alaskan Native		Total	
(%)	No.	(%)	No.	(%)	No.	(%)
(13.3)	2	(3.3)	0	(0.0)	60	(100.0)
(24.9)	1	(0.2)	2	(0.5)	429	(100.0)
(27.9)	0	(0.0)	0	(0.0)	140	(100.0)
(9.5)	0	(0.0)	0	(0.0)	21	(100.0)
(16.7)	0	(0.0)	0	(0.0)	6	(100.0)
(1.1)	0	(0.0)	0	(0.0)	92	(100.0)
(0.0)	0	(0.0)	0	(0.0)	2	(100.0)
(25.9)	1	(3.7)	0	(0.0)	27	(100.0)
(20.0)	0	(0.0)	0	(0.0)	20	(100.0)
(10.6)	0	(0.0)	1	(2.1)	47	(100.0)
(17.4)	0	(0.0)	1	(0.8)	132	(100.0)
(13.2)	0	(0.0)	0	(0.0)	38	(100.0)
(19.9)	**4**	**(0.4)**	**4**	**(0.4)**	**1,014**	**(100.0)**
(9.3)	Other**		1,151	(2.6)	43,864	(100.0)

eans of acquiring HIV infection are tabulated only in the category

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Plague in American Indians, 1956-1987

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INTRODUCTION

Bubonic plague (*Yersinia pestis* infection) is enzootic among native wild rodents and their fleas in much of the western United States from the Pacific coast eastward to about the 97th meridian (*1*). The disease in animals is characterized by sporadic or periodic epizootics among susceptible rodent populations, particularly members of the squirrel family (*Sciuridae*). Human cases acquired from wild rodents and their fleas result from chance encounters with plague-infected fleas or animals during epizootics, and cases typically occur as single, isolated events or in small clusters. In the past decade, approximately 19 cases per year have been reported. An apparently disproportionate number of these cases have occurred among American Indians (*2,3*). This report 1) presents information on the distribution of plague cases by racial/ethnic group and 2) discusses some of the factors believed to be responsible for the disparity.

METHODS

All human plague cases reported in the United States are confirmed at CDC and reported to the World Health Organization (*4*). Records of case investigations since 1956 are on file at CDC and include information on race and other pertinent epidemiologic data. In addition to human plague, animal plague has been monitored with some consistency since 1970 in collaboration with various state and federal agencies that report animal epizootics and submit animal specimens as a plague surveillance activity (*1*). Surveillance activities include serosurveys for antibodies to *Y. pestis* among wild carnivorous animals and, particularly on Indian reservations, among domestic dogs. Such surveys reflect plague infection among rodent populations and provide information on the distribution and abundance of plague and its geographic and temporal ebb and flow. These epidemiologic and surveillance files were reviewed in this study of plague and its frequency among American Indians.

RESULTS

Until 1965, when an outbreak of seven cases was reported from the Navajo Reservation in McKinley County, New Mexico, plague cases averaged one or two per year in the United States, and most of them occurred in the Pacific States (*5*). With the outbreak on the Navajo Reservation, plague cases began an increase that was centered in the southwestern states and that reached an average of 19 cases per year, with peaks of 40 cases in 1983 and 31 in 1984 (Figure 1).

FIGURE 1. Human plague cases among American Indians and others, by year, United States, 1956-1987

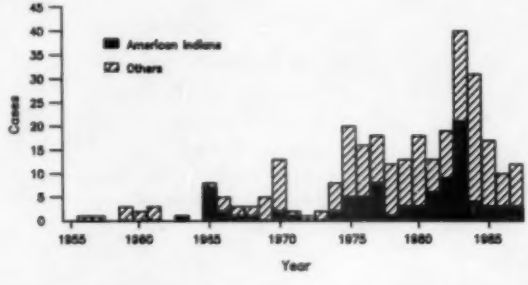
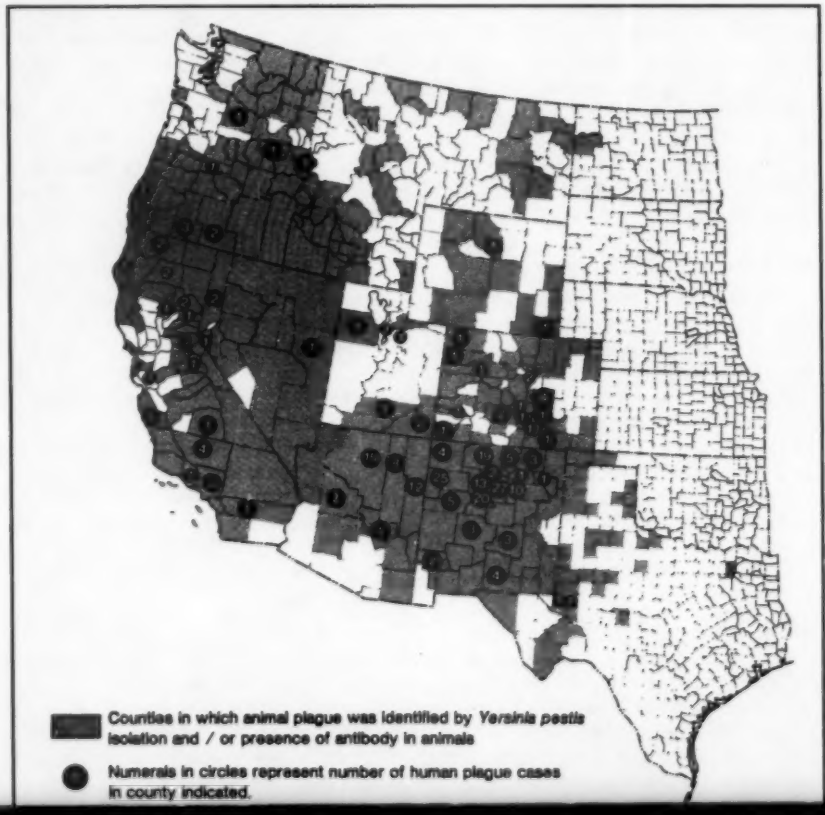


FIGURE 2. Geographic distribution of human and animal plague, United States, 1970-1986



Although plague in rodent populations is widely distributed and is reported frequently from 14 western states (Figure 2), most human cases are concentrated in the Southwest in an area that includes northern New Mexico, northeastern Arizona, and southern Colorado (Figure 2, Table 1). A similar concentration of cases is seen in California, southern Oregon, and western Nevada. In other areas, cases are infrequent or have never been reported. The center of distribution of human cases in the Southwest also is a center of distribution for American Indians and includes the 26,000-square-mile Navajo Reservation located in northwestern New Mexico, northeastern Arizona, and southernmost Utah.

In the period 1956-1987, 299 human plague cases were reported in the western United States. Most cases occurred in New Mexico (173 cases, 57.9%), followed by Arizona (43 cases, 14.4%), California (30 cases, 10.0%), Colorado (24 cases, 8.0%), and Oregon (10 cases, 3.3%). Scattered cases occurred in seven other western states, including Montana, which had its first case in 1987 (Table 1). The geographic distribution of cases from 1970 through 1986 and the distribution of animal plague detected by surveillance activities for the same period are shown in Figure 2.

Of the 299 persons who had plague in the study period, 91 (30.4%) were American Indians; 60 (20.0%) were Caucasian-Hispanics, mostly from north-central New Mexico; 146 (48.8%) were Caucasians from other states and New Mexico; one was Japanese; and one was Iranian. Of the 91 American Indians, 74 (81.3%) were Navajos and 12 (13.2%) were residents of various pueblos in north-central and western New Mexico. Cases also occurred on the Hopi, Mescalero Apache (New Mexico), Southern Ute (Colorado), and Warm Springs (eastern Oregon) reservations (Table 2). The attack rate among Navajos in 1983 was particularly high at 12.1/100,000 (*2*), when 19 cases were reported. During the same year, however, seven cases were reported from a predominantly Caucasian and Caucasian-Hispanic population in Santa Fe County in north-central New Mexico, for an attack rate of 9.3 (based on 1980 census figures for the entire county). Similarly, attack rates were 12.0 in Sandoval County in 1981 and

TABLE 1. Distribution of 299 human plague cases, by state, United States, 1956-1987

State	No. cases	(%)	No. fatal cases	(%)	CFR*
Arizona	43	(14.4)	6	(11.3)	14.0
California	30	(10.0)	7	(13.2)	23.3
Colorado	24	(8.0)	5	(9.4)	20.8
Idaho	2	(0.7)	1	(1.9)	50.0
Montana	1	(0.3)	0	(0.0)	0.0
Nevada	4	(1.3)	2	(3.8)	50.0
New Mexico	173	(57.9)	25	(47.2)	14.5
Oregon	10	(3.3)	4	(7.5)	40.0
Texas	2	(0.7)	1	(1.9)	50.0
Utah	7	(2.3)	2	(3.8)	28.6
Washington	1	(0.3)	0	(0.0)	0.0
Wyoming	2	(0.7)	0	(0.0)	0.0
TOTAL	**299**		**53**		**17.7**

*CFR = case-fatality rate.

10.3 in Rio Arriba County, both in 1975 and in 1976. If the population data for Santa Fe were adjusted to eliminate the urban population from the calculation, the attack rate obviously would be higher. The data show that the risk of plague infection among the racially mixed, largely Caucasian and Caucasian-Hispanic population in north-central New Mexico is at least as great as that among Navajos (Figure 1).

The distribution of Indian patients by age group and sex (Table 3) and case-fatality rates closely paralleled those for U.S. plague cases *in toto* (Table 1). Fifteen of the 91 Indian patients died, resulting in a case-fatality rate of 16.5%. In comparison, 53 (17.7%) of the total 299 patients in the United States died. Most Indian patients were young: 31 (34.1%) were under 9 years of age, 20 (21.2%) were ages 10-19 years, and 12 (13.2%) were ages 20-29. The case-fatality rate, however, was remarkably lower (9.6%) in the ≤9-year group than in older children and adults (20.0%). Distribution by sex was virtually even, with 47 male and 43 female patients. These figures closely parallel those for all U.S. plague cases, regardless of race; there is no evidence that

TABLE 2. Distribution of 299 human plague cases, by racial/ethnic group, United States, 1956-1987

Racial/ethnic group	No. cases	No. fatal cases
Caucasian	146	27
Caucasian-Hispanic	60	10
American Indian		
Navajo	74	14
Pueblo	12	1
Hopi	1	0
Mescalero Apache	2	0
Warm Springs	1	0
Southern Ute	1	0
Other*	2	1
TOTAL	**299**	**53**

*Other = 1 Japanese, died; 1 Iranian, survived.

TABLE 3. Plague cases among American Indians, by age group and sex, United States, 1956-1987

Age group (years)	Male		Female		Total	
	No. cases	No. fatal cases	No. cases	No. fatal cases	No. cases	No. fatal cases
≤9	16	2	15	1	31	3
10-19	13	3	7	1	20	4
20-29	6	3	6	1	12	4
30-39	4	0	3	0	7	0
40-49	4	0	3	0	7	0
50-59	2	1	2	0	4	1
60-69	2	1	5	1	7	2
≥70	0	0	3	1	3	1
TOTAL	**47**	**10**	**44**	**5**	**91**	**15**

American Indians are more susceptible than other races to infection, morbidity, or mortality from plague. In the instances in which the source of infection could be determined by epidemiologic investigation, no discernible difference was shown between cases among Indians and cases among other racial groups: available records for the period 1977-1986 showed that of the 62 cases among Indians, 41 (66%) were acquired via the bites of infective fleas, 14 (22%) resulted from direct contact with infected animals (rodents, rabbits, wild carnivores, or domestic cats), and 7 (11%) were of equivocal or unknown origin.

The number of plague cases in Navajo Indian populations each year appears to be proportionate to the scope and intensity of plague in animal populations. Plague surveillance on the Navajo Reservation includes annual spring surveys for plague antibodies among domestic dogs (*1*), an indirect measure of plague in rodents based on the fact that dogs become mildly infected, survive, and produce antibodies from ingesting infective rodent tissue. Data based on 680-1,400+ dog serum samples tested each year from 1983 through 1986 show that in 1983 (19 Navajo plague cases), 27.2% of all dogs on the Navajo Reservation that were tested had antibody titers of ≥32, and the geometric mean of positive titers (GMPT) was 247. In 1984 (four cases), 12.2% of dogs were seropositive, with a GMPT of 84; in 1985 (two cases), 4.2% were seropositive, with a GMPT of 58; and in 1986 (one case), 8.6% were seropositive, with a GMPT of 61. Although not directly comparable, surveillance data indicate that the data on dogs from the Navajo Reservation show a pattern of activity similar to that of human plague in the southwestern United States. The data indicate far greater plague activity in this region than similar serosurveys of dogs and wild carnivores have shown in other regions of the United States where plague is known to exist.

DISCUSSION

American Indians, principally Navajos, have a disproportionate share of the plague cases reported in the United States (Figure 2). For example, in 1981, 1982, and 1983, 46.2%, 47.4%, and 52.5%, respectively, of U.S. plague cases occurred among Indians (*5*). Similar attack rates, however, have occurred among Caucasian-Hispanics and Caucasians in adjoining north-central New Mexico, thus indicating that the high incidence of plague is a regional problem rather than a racial one. A more accurate statement might be that plague is more likely to occur among persons who live in rural and semirural locations in the plague-focus area of the Southwest than among persons in other parts of the country.

One explanation for the concentration of cases in the region is that plague in animals and vector fleas is more abundant and occurs more frequently in the plague-focus area of the Southwest than elsewhere, as suggested by data from serosurveys of persons with plague and of dogs living on American Indian Reservations. Unfortunately, the denominator data needed for testing the hypothesis are lacking; no comparable data are available on serosurveys from other regions.

A second explanation has to do with the presence, often in large numbers, of plague-susceptible rodents and vector fleas. Among these are rock squirrels (*Spermophilus variegatus*) and their fleas, *Diamanus montanus*. Squirrels have a predilection for habitats created by human activity and often live peridomestically. *Diamanus* fleas bite humans readily and transmit plague effectively. Together, they are directly responsible for at least 41% of human plague cases in the United States (*1*). Prairie dogs (*Cynomys gunnisoni*) and their fleas, *Opisocrostis hirsutus*, also are

widespread and abundant. Because of their abundance and susceptibility to plague, prairie dogs are a major amplifying host throughout the Southwest, including the Navajo Reservation. Prairie dog fleas are not inclined to bite humans, and most of the few cases acquired from prairie dogs follow the handling of a plague-infected animal, e.g., after preparing it as food. Human plague cases also have been acquired from various other hosts, including the antelope ground squirrel (*Ammospermophilus leucurus*) and its flea, *Thrassis bacchi*, resident in much of the Southwest.

PREVENTION AND CONTROL

Efforts to maintain surveillance and to conduct prevention and control programs in the plague-focus area of the Southwest, particularly on the Navajo Reservation, are made difficult by the immensity of the region and the dispersal of its people. Health education on how to recognize and avoid plague is a principal program element of the Indian Health Service (IHS) and Tribal authorities. The message is carried by IHS and Tribal Community Health Representatives by word of mouth, by the *Navajo Times*, and by Navajo-language radio and television programs. Insecticidal control of vector fleas is carried out by the IHS in response to human cases or the finding of plague in animal populations where there is potential for human exposure. Each spring on the Navajo Reservation, a systematic serosurvey for antibodies to *Y. pestis* in dog populations provides surveillance data and a degree of prediction concerning the course of plague activity during the summer transmission season.

In north-central New Mexico, where human populations are more concentrated, plague remains a problem in Indian pueblos and other semiurban areas. In such areas, new stategies for preventing human exposure are expected to be effective. These include the preemptive application of insecticides and rodenticides to control flea vectors and rodent reservoirs where risk of human exposure is known to be high. Field trials of permethrin, an insecticide, and cholecalciferol (Vitamin D3), a rodenticide, have shown that these materials are safe and effective when used against rock squirrels and their fleas in New Mexico residential areas and pueblos (*6*). Applications have been made to register both materials with the Environmental Protection Agency and appropriate state regulatory agencies. Permethrin is expected to be equally effective against other species of flea vectors and in other situations; however, on the immense and thinly populated Navajo Reservation, the use of rodenticides and preemptive control measures is not indicated. Using control measures for vector fleas in response to human cases and maintaining surveillance to detect plague in animal populations will continue to be the primary strategy.

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Leading Major Congenital Malformations Among Minority Groups in the United States, 1981-1986

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INTRODUCTION

Congenital malformations are a major cause of infant morbidity and mortality in the United States (*1*). In 1985, birth defects were the leading cause of infant mortality (*2*) and the fifth leading cause of years of potential life lost (*3*). Birth defects contribute significantly to chronic disease morbidity and related medical costs. Approximately 30% of all admissions to pediatric hospitals are associated with birth defects, and expenditures for medical care have been estimated at $1.4 billion per year (*4*).

All populations share the burden of congenital malformations, although the frequency and types of malformations may vary by race, ethnicity, and socioeconomic status (*5-11*). Access to medical care, nutrition, maternal lifestyles, and education are considered to be important factors in the occurrence of neural tube defects (*5*). Erickson (*6*) reported that in Atlanta, Georgia, the incidence of several common birth defects varies substantially between blacks and whites.

A large prospective study showed that blacks had higher overall rates of malformations than whites; this difference was primarily due to an increase in minor malformations such as polydactyly, branchial clefts, and supernumerary nipples, whereas whites had higher rates of major malformations and multiple malformations (*7*). Higher rates of neural tube defects were observed among infants of Hispanic women born in Puerto Rico and residing in Brooklyn than in their non-Hispanic black and white counterparts (*8*). American Indians appear to be at high risk for fetal alcohol syndrome. According to a recent study, the fetal alcohol syndrome rates for American Indians of the southwestern United States range from 13 to 103 per 10,000 live births (*9*). These rates vary greatly by subpopulation group (tribe). Several factors, such as culture influences, fertility, patterns of alcohol consumption, nutrition, and metabolic differences, are believed to play an important role in the distribution and occurrence of fetal alcohol syndrome among American Indians (*10*). Furthermore, oral clefts and congenital heart anomalies reportedly occur more frequently among American Indians in British Columbia than in the general population (*11*).

In 1981, CDC, through the Birth Defects Monitoring Program (BDMP) of the Birth Defects and Genetic Diseases Branch, Division of Birth Defects and Developmental Disabilities, Center for Environmental Health and Injury Control, began collecting data on the race and ethnicity of malformed infants born in the United States. This report summarizes available BDMP data on the leading major congenital malformations

among blacks, Hispanics, American Indians*, and Asians in the United States during the period 1981-1986 and compares the prevalence rates for these groups with those for whites.

MATERIALS AND METHODS

The primary objective of the BDMP, which was established at CDC in December 1974, is to serve as an early warning system for detecting changes or unusual trends in the rates of congenital anomalies in the United States. A secondary objective is to correlate occurrence patterns of birth defects with geographic and temporal trends in the distribution of possible human teratogens (*12*). The BDMP monitors 161 diagnoses, including structural, chromosomal, biochemical, and genetic disorders. BDMP data are obtained through the Commission on Professional and Hospital Activities (CPHA). The Commission's Professional Activities Study (PAS) system collects data from approximately 1,500 U.S. hospitals with obstetrics services. At each hospital, staff members of the medical records department review the chart of each newborn or stillborn delivered, prepare a case abstract, and send it to CPHA for processing. CPHA then sends portions of these abstracts to CDC for inclusion in the BDMP. Information provided to CDC includes the state, county, and hospital where the birth occurred; medical record number; zip code of maternal residence; birth date; birth status; gender; discharge status; and up to 15 diagnoses and 15 procedures coded according to the International Classification of Diseases, Ninth Revision. These data represent the largest source of information available on malformed infants born in the United States.

In 1981, the racial/ethnic variable was changed from "white or other" to more specific categories. The racial/ethnic classification now includes six categories: white, black, Hispanic, American Indian, Asian, and other. The infant's racial/ethnic classification is derived from the information recorded in the medical record. Children of mixed race/ethnicity are coded as "other." In addition, children of uncertain heritage are coded as "unknown."

During 1981-1986, approximately 1,236 PAS hospitals voluntarily participated in BDMP. The majority (69.2%) of these hospitals reported fewer than 1,000 births per year. The BDMP, through PAS, covered an average of 21.1% of all U.S. annual births.

In the period 1981-1986, the BDMP monitored 4,617,613 births. Information on specific race and ethnicity was available for 92.6% of these births. The BDMP coverage of the total number of births in the United States for each racial/ethnic group was as follows: 19.7% for whites, 16.4% for blacks, 17.5% for Hispanics, 9.3% for American Indians, and 8.3% for Asians. Rates of the 15 leading major congenital malformations for each racial/ethnic group were computed and simultaneously compared with those of the other groups (18 malformations overall) to determine which group had the highest rate. For each group, the total number of cases of each malformation was used as the numerator, and the total number of births reported through BDMP was used as the denominator. Later, rates of the 15 leading major malformations for each group were compared with the rates for whites in the BDMP for the same period. The rate ratio and its 95% confidence interval (CI) were computed. Statistical significance was established if the CI did not include one ($p<0.05$).

*Throughout this report, the term "American Indians" includes Alaskan Natives.

RESULTS

An analysis of the 18 major birth defects by racial/ethnic group showed that American Indians had the highest total rate, followed by whites, blacks, Asians, and Hispanics, respectively (Table 1). American Indians had the highest rates of hydrocephalus without spina bifida; atrial septal defect; valve stenosis and atresia; cleft palate without cleft lip; cleft lip with or without cleft palate; rectal atresia and stenosis; fetal alcohol syndrome; and autosomal abnormalities, excluding Down syndrome. Rates for clubfoot without central nervous system (CNS) defects, hip dislocation without CNS defects, and hypospadias were highest among whites. Rates for microcephalus, patent ductus arteriosus, and pulmonary artery stenosis were highest among blacks. The highest rates of anencephaly, spina bifida without anencephaly, and Down syndrome occurred among Hispanics. The rates for two major birth defects—anencephaly and ventricular septal defect—were highest among Asians.

TABLE 1. Rates of major congenital malformations, by race/ethnicity, United States, 1981-1986

	Rates†				
Malformation*	Blacks	Hispanics	American Indians	Asians	Whites
Anencephaly	2.1	4.4	3.6	4.4	3.0
Spina bifida without anencephaly	3.3	5.9	4.1	1.8	5.1
Hydrocephalus without spina bifida	8.1	4.6	10.8	4.8	5.4
Microcephalus	4.8	2.8	2.6	1.9	2.1
Ventricular septal defect	14.4	13.8	19.1	21.0	17.4
Atrial septal defect	2.1	1.2	4.1	2.5	2.1
Valve stenosis and atresia	5.9	1.9	8.2	2.8	3.2
Patent ductus arteriosus	49.9	20.7	33.5	25.1	26.5
Pulmonary artery stenosis	5.4	1.4	0	1.8	1.5
Cleft palate without cleft lip	3.7	3.7	9.8	4.6	5.9
Cleft lip with or without cleft palate	4.4	8.6	17.5	12.9	9.7
Clubfoot without CNS§ defects	19.9	19.1	15.5	14.4	27.5
Hip dislocation without CNS defects	13.8	24.0	31.4	25.0	32.3
Hypospadias	24.6	14.9	17.5	16.5	32.7
Rectal atresia and stenosis	2.8	3.0	4.6	3.8	3.7
Fetal alcohol syndrome	6.0	0.8	29.9	0.3	0.9
Down syndrome	6.5	11.6	6.7	11.3	8.5
Autosomal abnormalities, excluding Down syndrome	2.1	2.1	3.1	2.9	2.2
TOTAL	**179.9**	**144.4**	**222.0**	**157.8**	**189.8**

*By organ and/or system.
†Per 10,000 total births.
§Central nervous system.

Among blacks, rates for six malformations were statistically significantly higher and rates for nine malformations were statistically significantly lower than rates among whites (Table 2). Patent ductus arteriosus was the leading major congenital anomaly among blacks, possibly reflecting the high rate of prematurity in blacks (*3*). Patent ductus arteriosus occurred twice as frequently as hypospadias, the second leading major malformation among blacks. The fetal alcohol syndrome black:white rate ratio of 6.67 (CI = 5.71-7.79) was noteworthy.

Among Hispanics, the rates of anencephaly, microcephalus, and Down syndrome were significantly higher than those among whites (Table 3). Spina bifida without anencephaly was more prevalent among Hispanics, although the difference in rates was not statistically significant. Neural tube defects have been previously documented as being more prevalent among certain Hispanic subpopulations (*8*). The high rates of Down syndrome may be partially explained by advanced maternal age, inadequate access to medical care, or poor use of health services among Hispanics (*9*). On the other hand, rates for seven major congenital malformations were statistically significantly lower for Hispanics than for whites.

TABLE 2. Cases and rates of 15 leading major congenital malformations among blacks, by comparison with those among whites, United States, 1981-1986

Malformation	Blacks (N = 565,455)		Whites (N = 3,361,963)		Rate Ratio†
	Cases	Rate*	Cases	Rate	
Patent ductus arteriosus	2,822	49.9	8,916	26.5	1.88§
Hypospadias	1,393	24.6	10,995	32.7	0.75§
Clubfoot without CNS¶ defects	1,125	19.9	9,240	27.5	0.72§
Ventricular septal defect	815	14.4	5,854	17.4	0.83§
Hip dislocation without CNS defects	783	13.8	10,850	32.3	0.43§
Hydrocephalus without spina bifida	458	8.1	1,816	5.4	1.50§
Down syndrome	368	6.5	2,872	8.5	0.76§
Fetal alcohol syndrome	340	6.0	302	0.9	6.67§
Valve stenosis and atresia	336	5.9	1,060	3.2	1.84§
Pulmonary artery stenosis	308	5.4	489	1.5	3.60§
Microcephalus	273	4.8	706	2.1	2.29§
Cleft lip with or without cleft palate	248	4.4	3,270	9.7	0.45§
Cleft palate without cleft lip	211	3.7	1,991	5.9	0.63§
Spina bifida without anencephaly	184	3.3	1,716	5.1	0.65§
Rectal atresia and stenosis	156	2.8	1,252	3.7	0.76§

*Per 10,000 total births.
†Rate for blacks divided by the rate for whites.
§$p<0.05$.
¶Central nervous system.

As among blacks, patent ductus arteriosus was the most prevalent congenital malformation among American Indians; it was followed by hip dislocation without CNS defects and fetal alcohol syndrome (Table 4). The rate of fetal alcohol syndrome among American Indians was 29.9 per 10,000 total births, and the rate ratio was 33.22 (CI = 25.08-44.00) compared with whites. Rates for other malformations were also statistically significantly higher for American Indians than for whites; these malformations were valve stenosis and atresia, hydrocephalus without spina bifida, cleft lip with or without cleft palate, and cleft palate without cleft lip. Rates for hypospadias and clubfoot without CNS defects were statistically significantly lower for American Indians than for whites.

Most prevalent among Asians were patent ductus arteriosus and hip dislocation without CNS defects. Rates for anencephaly, Down syndrome, cleft lip with or without cleft palate, and ventricular septal defect tended to be statistically significantly higher among Asians than among whites. Rates for hip dislocation without CNS defects, hypospadias, and clubfoot without CNS defects were statistically significantly lower among Asians than among whites (Table 5).

TABLE 3. Cases and rates of 15 leading major congenital malformations among Hispanics, by comparison with those among whites, United States, 1981-1986

	Hispanics (N = 261,810)		Whites (N = 3,361,963)		
Malformation	Cases	Rate*	Cases	Rate	Rate Ratio†
Hip dislocation without CNS§ defects	628	24.0	10,850	32.3	0.74¶
Patent ductus arteriosus	542	20.7	8,916	26.5	0.78¶
Clubfoot without CNS defects	499	19.1	9,240	27.5	0.69¶
Hypospadias	390	14.9	10,995	32.7	0.46¶
Ventricular septal defect	360	13.8	5,854	17.4	0.79¶
Down syndrome	305	11.6	2,872	8.5	1.36¶
Cleft lip with or without cleft palate	226	8.6	3,270	9.7	0.89
Spina bifida without anencephaly	154	5.9	1,716	5.1	1.16
Hydrocephalus without spina bifida	120	4.6	1,816	5.4	0.85
Anencephaly	115	4.4	1,003	3.0	1.47¶
Cleft palate without cleft lip	98	3.7	1,991	5.9	0.63¶
Rectal atresia and stenosis	79	3.0	1,252	3.7	0.81
Microcephalus	73	2.8	706	2.2	1.33¶
Autosomal abnormalities, excluding Down syndrome	54	2.1	748	2.2	0.95
Valve stenosis and atresia	49	1.9	1,060	3.2	0.59¶

*Per 10,000 total births.
†Rate for Hispanics divided by the rate for whites.
§Central nervous system.
¶$p<0.05$.

DISCUSSION

The overall distribution of the leading major congenital malformations among blacks, Hispanics, American Indians, and Asians in the United States has not been previously documented. Results of several studies (*5-11*) have suggested that the rates of some malformations vary by socioeconomic status, race, and ethnicity. This report provides the first estimates of the 15 leading major congenital malformations among four minority groups in the United States and identifies groups at higher risk for some congenital anomalies. Furthermore, it provides baseline rates that should prove useful in identifying trends and clusters of malformations among minority groups.

The BDMP data provided in this report show that the frequency and the types of malformations vary greatly among minority groups in the United States. Certain racial/ethnic groups have a greater burden of some malformations than others. For blacks, the prevalence of patent ductus arteriosus is substantially higher than that for

Table 4. Cases and rates of 15 leading major congenital malformations among American Indians, by comparison with those among whites, United States, 1981-1986

Malformation	American Indians (N = 19,412)		Whites (N =3,361,963)		Rate Ratio†
	Cases	Rate*	Cases	Rate	
Patent ductus arteriosus	65	33.5	8,916	26.5	1.26
Hip dislocation without CNS§ defects	61	31.4	10,850	32.3	0.97
Fetal alcohol syndrome	58	29.9	302	0.9	33.22¶
Ventricular septal defect	37	19.1	5,854	17.4	1.10
Cleft lip with or without cleft palate	34	17.5	3,270	9.7	1.80¶
Hypospadias	34	17.5	10,995	32.7	0.54¶
Clubfoot without CNS defects	30	15.5	9,240	27.5	0.56¶
Hydrocephalus without spina bifida	21	10.8	1,816	5.4	2.00¶
Cleft palate without cleft lip	19	9.8	1,991	5.9	1.66¶
Valve stenosis and atresia	16	8.2	1,060	3.2	2.56¶
Down syndrome	13	6.7	2,872	8.5	0.79
Rectal atresia and stenosis	9	4.6	1,252	3.7	1.24
Atrial septal defect	8	4.1	718	2.1	1.95
Spina bifida without anencephaly	8	4.1	1,716	5.1	0.80
Anencephaly	7	3.6	1,003	3.0	1.20

*Per 10,000 total births.
†Rate for American Indians divided by the rate for whites.
§Central nervous system.
¶$p<0.05$.

any other group studied, and the rate of fetal alcohol syndrome is more than six times that for whites. The rates of neural tube defects and Down syndrome are significantly higher among Hispanics. American Indians have a 33-fold higher rate of fetal alcohol syndrome than whites, and they also have statistically significantly higher rates of oral clefts and most cardiovascular defects. The rate of anencephaly among Asians parallels that among Hispanics and is significantly higher than the rates among other groups. In addition, Asians have higher rates of Down syndrome, cleft lip with or without cleft palate, and ventricular septal defect.

Although minor birth defects were not included in this report because they are not a significant cause of morbidity and mortality, BDMP data showed that rates of polydactyly, breast anomalies (mostly supernumerary nipples), branchial clefts, and anomalies of the abdominal wall (mostly umbilical hernias) are highest among blacks.

The data in this report must be interpreted with caution. Some of the overall differences in the distribution of malformations among different racial/ethnic groups may be explained as follows: 1) In some instances, physicians may tend to look for a

TABLE 5. Cases and rates of 15 leading major congenital malformations among Asians, by comparison with those among whites, United States, 1981-1986

Malformation	Asians (N = 68,063)		Whites (N = 3,361,963)		Rate Ratio†
	Cases	Rate*	Cases	Rate	
Patent ductus arteriosus	171	25.1	8,916	26.5	0.95
Hip dislocation without CNS§ defects	170	25.0	10,850	32.3	0.77¶
Ventricular septal defect	143	21.0	5,854	17.4	1.21¶
Hypospadias	112	16.5	10,995	32.7	0.50¶
Clubfoot without CNS defects	98	14.4	9,240	27.5	0.52¶
Cleft lip with or without cleft palate	88	12.9	3,270	9.7	1.33¶
Down syndrome	77	11.3	2,872	8.5	1.33¶
Hydrocephalus without spina bifida	33	4.8	1,816	5.4	0.89
Cleft palate without cleft lip	31	4.6	1,991	5.9	0.78
Anencephaly	30	4.4	1,003	3.0	1.47¶
Rectal atresia and stenosis	26	3.8	1,252	3.7	1.03
Autosomal abnormalities, excluding Down syndrome	20	2.9	748	2.2	1.32
Valve stenosis and atresia	19	2.8	1,060	3.2	0.88
Atrial septal defect	17	2.5	718	2.1	1.19
Microcephalus	13	1.9	706	2.1	0.90

*Per 10,000 total births.
†Rate for Asians divided by the rate for whites.
§Central nervous system.
¶$p<0.05$.

particular malformation more in certain racial/ethnic groups than they would in others (detection bias). For example, some hospitals mainly serve minority groups believed to be at higher risk for some malformations. 2) Since BDMP data are not obtained from a random sample of U.S. hospitals, some of the participating hospitals may have a higher or lower than usual proportion of high-risk pregnancies and births. 3) Rates of congenital anomalies may be different in BDMP hospitals than in other hospitals because of their size or affiliation (public versus private). Nevertheless, hospitals that reported to BDMP in the study period ranged from those having fewer than 200 births to those having more than 1,000 births per year. The hospitals were located throughout the United States, and both privately owned and publicly owned hospitals were included. 4) Socioeconomic factors, such as education, access to medical care, nutrition, maternal lifestyles, and cultural influences, cannot be ruled out as major contributors to the burden of some malformations among certain minority groups. 5) Many of these major congenital malformations may have been associated with genetic and environmental factors. 6) The heterogeneity within minority groups is not addressed in this report. Some racial/ethnic subgroups (for example, certain American Indian tribes; Mexican-American, Cuban, or Puerto Rican Hispanics; and Japanese, Chinese, or other Asians) may account for a larger share of the group's total rate of some malformations.

In conclusion, the importance of monitoring congenital malformations by race and ethnicity has been demonstrated, and notable differences in the overall distribution of these anomalies have been identified. Much work remains to be done in the fight against the number one cause of infant mortality, and public health professionals need to address the problem of why certain minority groups have a higher risk for some congenital malformations than other segments of the population.

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Differences in Death Rates due to Injury Among Blacks and Whites, 1984

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INTRODUCTION

A 1985 report to Congress on "Injury in America" underscored the public health importance of injuries in the United States and indicated that the injury burden rests disproportionately among the poor (*1*), many of whom belong to minority groups. In a special report on minority health, the U.S. Department of Health and Human Services identified injuries as one of six problem areas that cause "excess deaths"* among blacks compared with whites (*2*). The other areas were cancer, heart disease and stroke, chemical dependency, diabetes, and infant mortality and low birth weight.

This summary describes the epidemiology of four causes of injuries with the largest disparities in black-white deaths in 1984 and discusses opportunities for intervention. It focuses on deaths from homicides, residential fires, drownings, and pedestrian mishaps—each of which accounted for over 1,000 deaths and showed a ratio of mortality rates between blacks and whites that exceeded 1.5. Other injuries, such as those caused by motor vehicles, accounted for many deaths but did not show statistically significant black-white differences in death rates; therefore, they are not included in this report.

METHODS

The numbers of deaths from the four injury categories selected for study were obtained from the mortality tapes of the National Center for Health Statistics (NCHS) for 1984, the latest year for which these data were available when this study was begun. Each death caused by injury was coded according to the Ninth Revision of the International Classification of Diseases, as shown in Table 1. Information on the NCHS tapes included the decedent's age, sex, race, and date of death. Death rates per 100,000 persons by race (blacks and whites only), sex, and age were calculated with the use of population estimates for 1984 from the U.S. Department of Commerce, Bureau of the Census (*3*).

RESULTS

In 1984, although blacks represented 12.0% of the U.S. population, they accounted for 15.3% of all deaths due to injury. Among the four causes studied for both blacks

*"Excess deaths" refers to the difference between the number of deaths actually observed among blacks and the number of deaths that would have occurred if blacks had died at the same rate, for each age and sex, as whites (*2*).

and whites, homicides accounted for the most deaths from injury (Table 1). For blacks, homicides were followed by pedestrian mishaps, residential fires, and drownings. For whites, homicides were followed by pedestrian mishaps, drownings, and residential fires.

The rate ratios of black-white deaths were highest for homicides (5.2:1), followed by residential fires (3.2:1), drownings (1.8:1), and pedestrian mishaps (1.6:1) (Table 1). Among the four race-sex groups, black males had the highest death rates from these causes; white females had the lowest death rates. Black females had death rates similar to those of white females except for homicides and residential fires; in those two categories, black females had higher death rates than both white females and white males.

Homicides accounted for the greatest disparity in injury mortality rates between blacks and whites. In 1984, the homicide rate for blacks was 29.0 per 100,000. This rate was just over five times higher than the homicide rate for whites of 5.5 per 100,000 (Table 1). For both blacks and whites, the age-specific homicide rates decreased from infancy to ages 5-9 years and then began to increase (Figure 1). For blacks, the rates peaked at ages 25-29 years, declined markedly to ages 65-69, and then declined slightly. In contrast, the rates for whites peaked at ages 20-24, declined gradually to ages 65-69, and then slightly increased.

For both males and females in 1984, homicide rates were highest among young adults ages 20-39. Rates for black males peaked at 99.6 for the 25- through 29-year-old age group. Although rates were higher for black males than for any other race-sex group, black females also were at high risk of homicide. Their rate also peaked in the 25- through 29-year-old age group at 21.9. Rates for black males were four to five times greater than those for black females in all age groups above 15 years of age. A similar pattern was seen among whites—that is, young adults were at highest risk, and males had consistently higher rates than females in each age group. The ratio of male-to-female rates, however, was less for whites, with the rate for white males

TABLE 1. Selected fatalities resulting from injury, by race and sex, United States, 1984

Cause (external code)	Sex	Black		White		Rate ratio
		Rate	(Deaths)	Rate	(Deaths)	
Homicide	Male	48.7	(6,563)	8.3	(8,171)	5.9
(E960-E969)	Female	11.2	(1,677)	2.9	(2,956)	3.9
	Total	29.0	(8,240)	5.5	(11,127)	5.2
Residential fire	Male	6.1	(818)	1.9	(1,832)	3.3
(E890-E899,	Female	3.7	(550)	1.1	(1,165)	3.2
Location = 0)	Total	4.8	(1,368)	1.5	(2,997)	3.2
Drowning	Male	6.8	(911)	3.4	(3,347)	2.0
(E830,E832,E910)	Female	0.9	(137)	0.8	(790)	1.2
	Total	3.7	(1,048)	2.1	(4,137)	1.8
Pedestrian	Male	7.7	(1,044)	4.3	(4,237)	1.8
(E810-E825,	Female	2.2	(325)	1.8	(1,834)	1.2
Person = 0.7)	Total	4.8	(1,369)	3.0	(6,071)	1.6

*Deaths per 100,000.

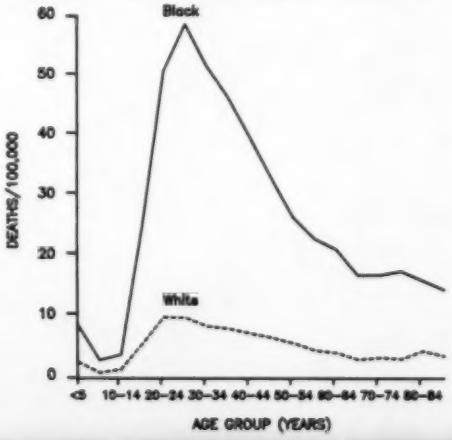
generally ranging from two to three times higher than that for white females. Among blacks, 61.7% of homicides were committed with firearms, compared with 58.6% among whites.

For residential fires, black males had almost twice the death rate of black females, over three times the rate of white males, and over five times the rate of white females (Table 1). The distributions of age-specific death rates between the races, however, were similar (Figure 2). Black and white children (0-4 years) and elderly persons (>65 years) had the highest death rates due to residential fires. The rates for blacks and whites declined after ages 0-4 years, but after ages 40-44 years the rates increased more rapidly for blacks than for whites. Although blacks consistently had higher death rates due to residential fires than whites, the smallest rate differences were at ages 10-29 years, and the greatest were at ages 0-4 and 70+ years (Figure 2).

For drownings, black males had twice the death rate of white males, over seven times the rate of black females, and over eight times the rate of white females (Table 1). Blacks in the age groups 0-4 and 70+ years had lower rates than whites of the same age. In all other age groups, blacks had higher rates (Figure 3). For both races, the death rates peaked at ages 15-19 years and then declined with increasing age. By ages 70-74 years, the death rate among blacks fell to a level below that of whites.

For pedestrian mishaps, black males had nearly twice the death rate of white males, more than three times the death rate of black females, and over four times the rate of white females (Table 1). Blacks ages 15-19 years had a lower rate than whites of the same age (1.9 versus 3.0) (Figure 4). At all other ages, blacks had higher rates than whites. The rates for blacks and whites declined from infancy to ages 10-14 years. This decline began at an older age group for black children (5-9 years) than for white children (0-4 years) and ended at an older age for blacks (15-19 years) than for whites (10-14 years).

FIGURE 1. Homicide rates, by age group and race, United States, 1984



DISCUSSION

Overall, death rates resulting from homicides, residential fires, drownings, and pedestrian mishaps were higher among blacks than among whites. With few exceptions, this increased risk among blacks occurred over the entire age range. Black females generally had higher death rates than white females, and black males generally had the highest death rates. Of the four types of injuries studied for both

FIGURE 2. Death rates due to residential fires, by age group and race, United States,1984

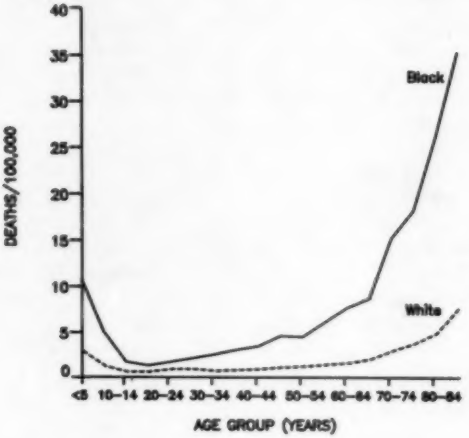
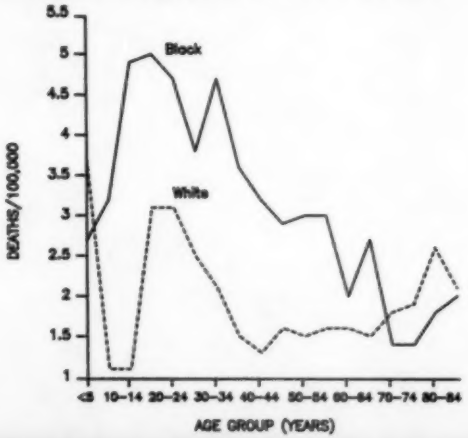


FIGURE 3. Drowning rates, by age group and race, United States, 1984



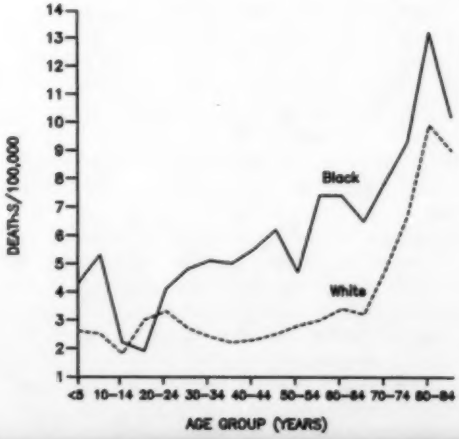
blacks and whites, homicide was the leading cause of death; residential fires ranked second for blacks and fourth for whites.

Reasons are unclear as to why blacks have higher death rates from these injuries than whites, because little information is available on race-specific risk factors. Previous studies have shown that deaths from homicides, residential fires, drownings, and pedestrian mishaps have common risk factors such as low socioeconomic status (SES) and alcohol consumption (*1,2,4-7*). Each cause, however, also has unique risk factors associated with death.

Clearly, blacks have had dramatically higher homicide rates than whites. Various explanations for this racial difference have been considered. Poverty has been suggested as an underlying factor in homicide (*5,6*), and indeed poverty is more prevalent among blacks than among whites in this country (*8*). Supporting the suggestion that poverty increases the risk of homicide, research has shown that racial differences in homicide rates all but disappear when SES status is taken into account (*9,10*). These studies suggest that interventions should not be targeted to blacks as a race but rather to the impoverished. Even if low SES were found unequivocally to increase the risk of homicide, however, the mechanism by which it increases that risk would need to be identified.

Other data show a commonality in homicide patterns among blacks and whites which suggests that, despite dramatic differences in the risk of victimization, the fundamental causes of homicide may be much the same regardless of race. For both races, males were at higher risk of death from homicide than females, perhaps because of the way males are taught to respond to conflicts. Accordingly, intervention efforts might focus on teaching appropriate methods for resolving conflicts. Profound questions remain to be answered concerning risk factors related to homicide, such as drug and alcohol abuse and ready accessibility of firearms. A multidisciplinary

FIGURE 4. Death rates due to pedestrian mishaps, by age group and race, United States, 1984



approach will be needed to define these risk factors and to develop and implement successful intervention strategies.

For residential fires, the risk factors for a fatal outcome include a lack of smoke detectors, physical conditions that prevent escape from a fire, inappropriate heating devices, residence in poor housing, smoking in bed, and decreased availability and slower response of fire department services (*4,11-13*). Blacks are less likely to own smoke detectors than whites (*11*). Moreover, since blacks are disproportionately poorer than whites (31.1% of blacks were below the poverty level in 1986, compared with 10.2% of whites) (*8*), they may have greater exposure to other risk factors, such as living in poor housing and using inappropriate heating devices more frequently than whites. Aside from these factors, no conclusive evidence links risk factors specifically to race.

For drowning, the risk factors include living in a warm climate, swimming in undesignated areas, inability to swim, not using or misusing personal flotation devices, and using open boats (*4,14,15*). The distribution of these risk factors among blacks is not clearly understood, because specific research has not been done. Because of income disparity among whites and blacks, however, fewer blacks than whites may own swimming pools. Consequently, the relatively lower drowning rate among black children compared with white children may result from less exposure to swimming pools. The higher risk among blacks 15-19 years of age may be related to lack of instruction in swimming and to recreation at less guarded bodies of water, such as lakes, rivers, and ponds (*16*).

For pedestrian mishaps, specific reasons for the higher death rate among blacks have not been identified; however, epidemiologic studies have suggested that fatal pedestrian mishaps are associated with vehicle size and design, speed of impact, contact point, and behavior of the pedestrian and driver (*4,17*). One hypothesis is that blacks may walk more often than whites because a smaller percentage of them own motor vehicles. Death rates from pedestrian mishaps reportedly have been highest in the Southeast and Southwest regions of the United States, and—in contrast to the death rate for whites—the highest death rates for blacks have occurred in rural areas (*4,17*). By one estimate, the ratio of death to injury in rural areas was three times that in urban areas (*4*). More blacks may live in rural areas, where driving rules may be less strictly enforced than in urban areas. Thus, impact speeds between vehicles and pedestrians may be greater, increasing the likelihood of a fatal outcome. Delayed or inadequate emergency medical services in rural areas may also play a role.

Opportunities exist to reduce the death toll from homicides, residential fires, drownings, and pedestrian mishaps, although a complete knowledge of race- and cause-specific risk factors is lacking. Interventions may be aimed either at known risk factors common to these injuries or at high-risk groups. Although these approaches are not mutually exclusive, those aimed at common risk factors may be preferable. If successful, such approaches may bring about gains in several areas. For example, an intervention aimed at improving SES in a population might also decrease the number of deaths from homicides, residential fires, and pedestrian mishaps. A drawback to this approach, however, is that no one knows what specific aspects of low SES are associated with excess deaths. On the other hand, interventions aimed at high-risk groups are more focused because the control efforts are applied more selectively. For example, a program that promotes the installation and maintenance of smoke detectors may target children and the elderly, the highest risk groups for deaths in

residential fires. Another program offering swimming instructions and water safety skills may target black males 15-19 years of age, the highest risk group for drownings.

In conclusion, fatal injuries exact a disproportionate burden on blacks compared with whites. Homicides, residential fires, drownings, and pedestrian mishaps account for most of this difference. To reduce the higher death rates due to injury among blacks, investigators must define the race- and cause-specific risk factors that can be used to guide intervention strategies. These findings then must be integrated into public health programs designed to reduce the injury burden on blacks in this country.

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Dental Caries and Periodontal Disease Among Mexican-American Children from Five Southwestern States, 1982-1983*

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INTRODUCTION

The Hispanic population may be the largest minority group in the United States by the end of the 20th century. The 1980 census showed an estimated 14.6 million Hispanics in the United States (*1*), an increase of about 5.6 million (62%) since 1970, although even this figure may be an underestimate.

Despite the growing numbers of Hispanics, little information is available about their health status (*2,3*). Because of the paucity of data, Public Law 94-311, enacted in 1976, required designated federal agencies to collect, analyze, and publish health, social, and economic data on Hispanic Americans. In response to this mandate from Congress, and following the recommendations of the expert committee of the National Academy of Public Administration (*4*), the National Center for Health Statistics (NCHS) organized a health and nutrition survey of persons between the

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ages of 6 months and 74 years who were of Hispanic descent and who lived in selected geographic areas of the United States. This survey is known as the Hispanic Health and Nutrition Examination Survey (HHANES). Persons were considered eligible to participate in the survey on the basis of self-reported ethnicity or national origin. HHANES was conducted in the period 1982-1984 and included Hispanics residing in three geographic regions of the United States: selected counties in five southwestern states (Texas, New Mexico, Arizona, Colorado, and California), the Miami area (Dade County), and selected counties or areas of New York City, New Jersey, and Connecticut. The three components of HHANES targeted Hispanics of Mexican, Cuban, and Puerto Rican origin, respectively. These subpopulations represent the majority of Hispanics in the United States (*1*). Data were collected in the Southwest from 1982 through 1983, and data collection was completed in Miami and New York City in 1984.

This paper describes the estimated prevalence of dental caries and periodontal disease among Mexican-American children 5-17 years of age living in the five selected southwestern states. The prevalence was based on data obtained from dental examinations of 2,550 children, including 22 children who lived in the selected households but who were not Mexican-Americans.

BACKGROUND

Little information is available on the oral health of Hispanic Americans. Previous surveys conducted by NCHS and the National Institute of Dental Research (NIDR) have included few Americans of Hispanic descent (*5-7*). In the first National Health and Nutrition Examination Survey (NHANES I), for example, only about 700 persons reported that they were of Mexican ancestry.

The only extensive survey that included a large sample of Hispanics was the Ten-State Nutrition Survey of 1968-1970 (*8*), which showed that Hispanic children in Texas (6-17 years of age) had more missing teeth and higher scores based on decayed, missing, and filled teeth (DMFT) than non-Hispanic white and black children. By contrast, Hispanic children in the nine other states had lower mean DMFT scores than either non-Hispanic whites or blacks residing in these states. In Texas, fewer Hispanics had gingivitis than blacks or non-Hispanic whites, whereas in the other states the prevalence of periodontal disease among Hispanic children was similar to that of non-Hispanic white children but lower than that of black children.

In the 1978-1981 National Preventive Dentistry Demonstration Program (*9,10*), Hispanic children 6, 8, 10, and 12 years of age from three cities where water supplies are fluoridated—New York; El Paso, Texas; and Hayward, California—participated in the study. Mean scores based on decayed, missing, and filled permanent tooth surfaces (DMFS) of El Paso Hispanic children were less than half those of Hispanic children from the two other cities, although they were similar to the scores of El Paso's non-Hispanic children.

Several smaller surveys of Hispanic or Mexican migrant workers have also been reported (*11-13*). In Michigan, children of Hispanic migrant workers had a higher percentage of unmet dental needs than U.S. children overall in 1971-1974 (*11*). A similar conclusion was reached by other investigators (*12,13*). Many Hispanic Americans, however, live under different social and environmental conditions than do Hispanic migrant workers, thus the general applicability of these studies may be limited.

Health attitudes and behaviors of Hispanic Americans reportedly differ from those of non-Hispanic Americans (*14,15*). Hispanics have been found to use professional dental services infrequently, and many of them consult a dentist only when they are in pain (*14*). Differences also have been found between Hispanics and non-Hispanics in the reasons for and frequency of dental visits and in the use of cosmetic, orthodontic, surgical, and prosthetic services. These differences remain even after income and education have been considered.

Because of limited data on the dental health of Hispanic children, investigators have had difficulty in reaching firm conclusions. The data do suggest, however, that the prevalence of dental caries among Hispanics does not differ from that of the population at large in the same geographic region.

METHODS

HHANES Survey Design

The HHANES sample design was a four-stage cluster sample selected with probabilities proportional to size. The four stages were 1) primary sampling units (PSUs), made up of counties or small groups of contiguous counties, 2) segments (clusters of households), 3) households, and 4) eligible persons. A detailed description of the sample design is presented elsewhere (*4*); a summary description is presented here.

During the planning phase of HHANES, criteria were formulated that specified the eligibility requirements for inclusion in HHANES. A county was included in the HHANES sampling frame if it satisfied at least one of the following conditions: 1) the county's Hispanic-origin population numbered at least 30,000, 2) the county's Hispanic-origin population was at least 10,000 but less than 30,000, and it constituted at least 5% of the total county population, 3) the county's Hispanic-origin population was at least 5,000 but less than 10,000, and it constituted at least 10% of the total county population, or 4) the county's Hispanic-origin population was less than 5,000, and it constituted at least 15% of the total county population.

By design, therefore, HHANES is not a representative national survey of all Hispanics residing in the United States, but rather it is representative of Hispanics living in the areas sampled. These areas include approximately 76% of all Hispanics living in the United States (*4*). The southwestern portion of HHANES covered approximately 97% of all Mexican-Americans within its sampling frame (five selected states).

In the Southwest, 14 PSUs were randomly selected from the 193 located in the five southwestern states. The selection of these PSUs took into consideration the number of Mexican-Americans, the ratio of 1980 to 1970 Mexican-American population, the median income, and the percentage of urban residents. Households in the 14 selected counties were divided into segments; the minimum number of Mexican-Americans in each segment ranged from 50 to 100 persons. Segments that had fewer than 50 Mexican-Americans were excluded from the sample. The eligible segments were stratified by density of Mexican-American population and economic status, as determined by rent paid or value of homes. Segments were selected with equal probabilities within each of the 14 selected PSUs. From each of these segments, six households were then selected. Mexican-Americans residing in each selected household were sampled by using the following probabilities: three-fourths of eligible

Mexican-Americans ages 6 months to 19 years, one-half of eligible Mexican-Americans ages 20 to 44 years, and all eligible Mexican-Americans ages 45 to 74 years. By design, not all persons included in the sample underwent the same interviews in their residences nor the same medical, dental, and biochemical assessments at the mobile examination center. After being informed of their inclusion in the survey, some persons could not or would not participate in the interviews, and some of the persons interviewed could not or would not be examined. A total of 7,240 persons received dental examinations (Table 1).

Oral Conditions Measured in HHANES

In HHANES, plans were made for each participant to receive a medical and dental examination and to provide a 24-hour dietary-recall record during an interview conducted by a trained dietary interviewer. Other demographic and health-related behaviors were also to be recorded. Questions of relevance to dentistry included reasons for and frequency of dental visits, preventive health behaviors, coverage by dental insurance, and an evaluation of perceived oral health.

Dental examiners measured the prevalence of dental caries by using NIDR criteria (*7*), periodontal disease by using the Periodontal Index (PI) (*16*), oral hygiene status by using Debris (DI) and Calculus (CI) Indexes (*17*), and malocclusion status and history of orthodontic treatments as defined by NIDR (*18*). The examiners also evaluated the denture status of partially and completely toothless examinees, and they estimated the need for restorative care by using the NIDR Dental Restorative Treatment Need Index (*18*). The dental examiners were all trained by NIDR staff.

Statistical Analysis

When dental data are abstracted from national surveys, analysts should take into consideration the complexity of the sample design (clustering) and the disproportionate probabilities of selection (sampling weights) of the persons in the sample.

TABLE 1. Sample sizes for the southwestern portion of HHANES, by age group, 1982-1983

Age group (years)	Total sample size	Sample interviewed	Sample examined	Sample in dental analysis
6 mo-4 yr	1,238	1,142	1,029	1,010
5-9	1,299	1,189	1,029	1,082
10-11	489	450	417	411
12-19	1,755	1,555	1,357	1,346
20-24	759	644	534	514
25-34	1,423	1,233	1,046	989
35-44	883	746	641	612
45-54	1,029	792	668	638
55-64	699	540	450	438
65-74	320	263	216	200
Total	**9,864**	**8,554**	**7,462**	**7,240**

Source: National Center for Health Statistics, CDC.

Failure to adjust for the clustering effects may lead to an underestimation of the standard errors of the computed statistics and may result in higher probabilities of detecting statistically significant differences when no such differences exist (type I error) (*19,20*). Sampling weights permit results to be generalized to the population from which the sample was drawn (Mexican-Americans in the five selected southwestern states).

In the analyses presented in this paper, sampling weights were used and clustering effects were accounted for. Programs of the Organized Sets of Integrated Routines in Statistics (*21*), supported by the Institute for Survey Research, University of Michigan, were used to compute weighted means, regression coefficients, and percentages. So that the clustering effects introduced by the sample design would be accounted for, standard errors—computed for a simple random sample—were multiplied by their design effects. The design effects represent the estimated increase in the variance of the variable under study (for example, DMFS scores) that has resulted from selecting participants in clusters rather than individually, as when a simple random sample design is used. The design effects were computed for the purpose of this analysis for the DMFT, DMFS, PI, DI, and CI scores (Table 2). Each of these variables was stratified by age, income, and gender, and design effects were computed for each of the cells in the age-gender-income matrix. Design effects larger than one were added and their mean was computed for estimating an overall design effect that was used to adjust the standard errors of each variable. Testing for statistical significance between means or percentages was based on the comparison of 95.0%, 99.0%, or 99.9% confidence intervals.

RESULTS

Dental Caries by Age, Gender, Income, and Type of Tooth Surface

Of all the Mexican-American children between 5 and 17 years of age residing in the Southwest, 46.0% were free of caries (Table 3). Overall, 4.4% had DMFT scores higher than nine. The percentage of children with DMFT scores of nine or above ranged from 1.0% (for 10-year-olds) to 19.4% (for 17-year-olds).

The distribution of the mean DMFS by age is presented in Figure 1. The main contributor to the DMFS after age 8 was filled tooth surfaces; the percentage of filled

TABLE 2. Design effects used to adjust the standard errors computed by using a simple random sample design

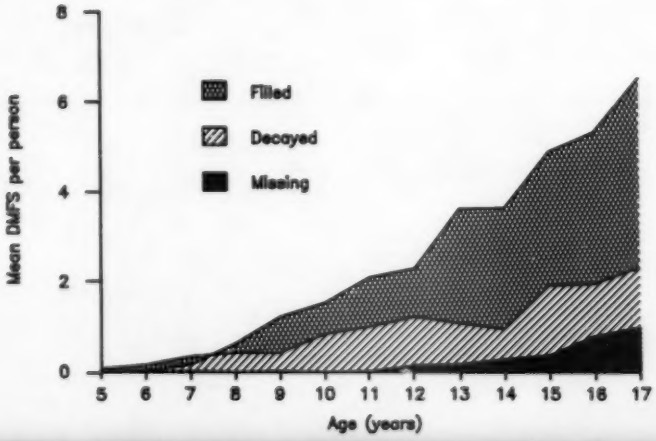
Variable	Design effect*
DMFT†	1.45
DMFS§	1.52
PI¶	1.25
DI**	1.49
CI††	1.18

*See "Statistical Analysis" section for description.
†DMFT = Decayed, missing, and filled teeth.
§DMFS = Decayed, missing, and filled tooth surfaces.
¶PI = Periodontal Index (*16*).
**DI = Debris Index (*17*).
††CI = Calculus Index (*17*).

TABLE 3. Percent distribution of Mexican-American children 5-17 years of age in Southwestern HHANES, by age and number of decayed, missing, and filled teeth

Age (years)	Number of decayed, missing, and filled teeth			
	0	1-4	5-8	9-28
5	97.3	2.7	0.0	0.0
6	92.2	7.8	0.0	0.0
7	71.9	28.1	0.0	0.0
8	64.0	35.7	0.3	0.0
9	56.7	42.4	0.9	0.0
10	47.8	48.4	2.7	1.0
11	40.0	51.7	7.5	0.7
12	29.7	54.6	14.5	1.3
13	22.3	47.1	23.9	6.7
14	25.7	48.9	21.7	3.7
15	20.2	37.0	34.5	8.3
16	18.3	38.7	26.7	16.3
17	14.6	35.1	30.9	19.4
All	**46.2**	**36.8**	**12.6**	**4.4**

FIGURE 1. Mean number of decayed, missing, and filled permanent tooth surfaces (DMFS) among Mexican-American children, by age, Southwestern HHANES, 1982-1983



to total DMFS surfaces was 66.0% for all Mexican-Americans in the Southwest. The rate of increase in number of filled surfaces with increasing age was three times larger than the rate of increase in number of decayed surfaces (regression slopes for the number of filled and decayed surfaces with increasing age were 0.54 and 0.18, respectively). The number of missing teeth was significantly lower than the number of decayed or filled teeth ($p<0.001$).

Mexican-American females had a significantly higher mean number of filled occlusal surfaces than males ($p<0.01$), although no significant difference was detected between males and females in decayed and missing occlusal and other tooth surfaces (Table 4). Occlusal tooth surfaces of molars were the most susceptible to decay, and anterior tooth surfaces were the least decayed.

Dental Caries by Income Status

No significant association was shown between mean DMFS scores and income. However, the percentages of decayed or filled tooth surfaces, out of the total DMFS scores, were significantly different between low-income families (less than $6,000 annual income) and high-income families ($40,000 or more annual income) (Table 5).

TABLE 4. Mean number of decayed, missing, and filled tooth surfaces among Mexican-American children 5-17 years of age, by sex and type of tooth surface, Southwestern HHANES, 1982-1983

	Male		Female	
Tooth surface	Mean	SEM*	Mean	SEM
Posterior teeth				
Occlusal surfaces				
Decayed	0.51	0.04	0.56	0.04
Filled	1.10	0.07	1.53	0.09
Missing	0.04	0.01	0.04	0.01
BL† surfaces				
Decayed	0.31	0.03	0.26	0.02
Filled	0.74	0.04	0.87	0.05
Missing	0.08	0.00	0.08	0.01
MD§ surfaces				
Decayed	0.07	0.01	0.08	0.01
Filled	0.20	0.03	0.27	0.03
Missing	0.08	0.01	0.08	0.01
Anterior teeth				
BL surfaces				
Decayed	0.05	0.02	0.03	0.01
Filled	0.05	0.01	0.06	0.01
Missing	0.00	0.00	0.00	0.00
MD surfaces				
Decayed	0.06	0.02	0.05	0.01
Filled	0.03	0.00	0.05	0.01
Missing	0.00	0.00	0.00	0.00

*SEM = Standard error of the mean.
†BL = Buccal and lingual surfaces.
§MD = Mesial and distal surfaces.

Children from low-income families had a higher percentage of tooth surfaces that were decayed and less that were filled than children from high-income families ($p<0.05$).

Comparison of Results from HHANES, NIDR, and NHANES I Surveys

Figure 2 presents a comparison of the mean DMFS of Mexican-American children 5 through 17 years of age in Southwestern HHANES (1982-1984), children residing in the western states* during NHANES I (1971-1974), and those residing in Region V† of the NIDR survey (1979-1980). Except for children ages 13 and 17, the mean DMFS scores for Mexican-American children in 1982-1983 did not differ significantly from mean DMFS scores of children from Region V in the NIDR survey, whereas the mean DMFS scores of children examined during NHANES I were significantly higher after age 9 ($p<0.05$).

Periodontal Disease and Oral Hygiene

The prevalence of periodontal disease, plaque, and calculus in Mexican-American children 5-17 years of age is presented in Tables 6 and 7. A high proportion (76.9%) of Mexican-American children suffer from gingivitis. The Mexican-American children in 1982-1983 had significantly higher mean DI and PI scores than children from the western states examined during NHANES I even after adjustment was made for income in 1971-1974 and 1982-1983. Mexican-American children from high-income families had lower PI, CI, and DI scores (approximately 40%, 70%, and 15%, respectively) than Mexican-American children from low-income families.

A significantly higher percentage of Mexican-American children had gingivitis in 1982-1983 than children (all races) residing in the western states in 1971-1974 (Table 7). The mean number of teeth with mild gingivitis among Mexican-Americans ages 5-17 years was 6.14, whereas the mean number of teeth with more severe forms of gingivitis was 1.01. Four children between the ages of 5 and 17 years had periodontal pockets.

*Arizona, California, Colorado, Idaho, Kansas, Montana, Nebraska, Nevada, New Mexico, North Dakota, Oklahoma, Oregon, South Dakota, Texas, Utah, Washington, and Wyoming.
†Arizona, Colorado, New Mexico, and Texas.

TABLE 5. Mean DMFS* scores and percent of total DMFS among Mexican-Americans 5-17 years of age, by family's annual income, Southwestern HHANES, 1982-1983

Family income in U.S. dollars	n†	Mean DMFS	Percent of Total DMFS		
			Decayed	Filled	Missing
<6,000	292	4.4	33.4	63.5	3.1
6,000-9,999	409	3.4	29.5	62.8	7.7
10,000-14,999	440	3.2	30.7	63.8	5.5
15,000-24,999	665	3.7	26.4	67.5	6.1
25,000-39,999	403	3.8	21.6	71.3	7.1
40,000+	113	4.2	15.9	79.1	5.0

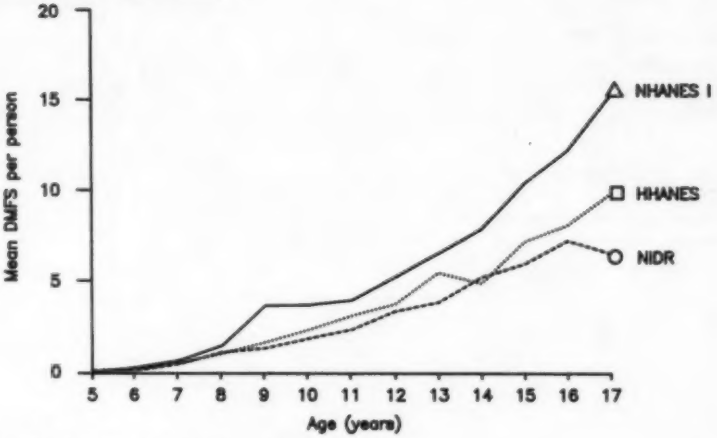
*Decayed, missing, and filled permanent tooth surfaces.
†n = number of persons. The total number of children who had dental examinations and for whom income information was available was 2,322.

DISCUSSION

Survey Design

Planning and conducting a survey like HHANES presents some difficult choices. The reference population mainly resides in a few states (*3*), with a relatively small portion of the population residing in the rest of the country. Because of this distribution of Mexican-Americans, the survey was limited to areas where the majority of Mexican-Americans reside. The costs of including every Mexican-American in the sampling frame would be prohibitive; therefore, the geographic area included in HHANES was restricted to U.S. counties having a large (as described in the section titled HHANES Survey Design) Mexican-American population. As a

FIGURE 2. Mean DMFS scores of Mexican-American children in Southwestern HHANES (1982-1983), western states of NHANES I (1971-1974), and Region V of NIDR (1979-1980), by age and survey*



*See text for definitions and explanations.

TABLE 6. Comparison of index scores of Mexican-American children 5-17 years of age who participated in Southwestern HHANES, 1982-1983, with those of participants in the western states* of NHANES I, 1971-1974, by gender

	HHANES (1982-1983)				NHANES I (1971-1974)			
	Male	SEM†	Female	SEM	Male	SEM	Female	SEM
DI§	0.84	0.01	0.75	0.01	0.68	0.03	0.53	0.02
CI¶	0.10	0.01	0.08	0.01	0.01	0.02	0.09	0.01
PI**	0.34	0.01	0.29	0.01	0.22	0.02	0.14	0.02

*For list of 17 states, see text.
†Standard error of the mean.
§Debris Index (*17*).
¶Calculus Index (*17*).
**Periodontal Index (*16*).

result, the southwestern portion of HHANES cannot be considered a national survey of all Mexican-American residents but rather of approximately 84% of them, and of 97% of the 1980 Mexican-origin population in the Southwest (*4*).

In the analysis of data from a complex survey like HHANES, a number of factors need to be considered. The available algorithms used for computing means and their standard errors assume that a proportionate simple random sample is selected. For complex survey designs, this assumption is invalid, and adjustment for the sampling weights and clustering of individuals introduced by the previously described sampling strategy should be made in order to reach more accurate and precise population estimates (*20*). (An accurate estimate is an estimate with minimum total error that includes bias as well as the variance, whereas a precise estimate is an estimate with minimum variance.) Further, the sample weights provided by NCHS also include adjustments for nonresponse and noncoverage that were made to compensate for nonrepresentation of some of the targeted population. Analyses presented in this paper have accounted for both clustering and sampling weights.

Comparison with Other Surveys

Analytical considerations create a problem when the results of one national survey are compared with those of another. The NHANES I, the NIDR Dental Caries Prevalence Survey, and the Southwestern HHANES were conducted at different times

TABLE 7. Comparison of periodontal disease status of Hispanic participants 5-17 years of age in Southwestern HHANES, 1982-1983, with those of participants in the western states* of NHANES I, 1971-1974, by age

Age (years)	Percent of individuals					
	HHANES			NHANES I		
	No disease	Gingivitis	Pockets	No disease	Gingivitis	Pockets
5	60.4	39.6	0.0	100.0	0.0	0.0
6	38.0	61.4	0.6	98.5	1.5	0.0
7	24.4	75.6	0.0	92.1	7.9	0.0
8	16.7	83.3	0.0	85.2	12.9	1.9
9	16.9	83.1	0.0	83.3	16.7	0.0
10	16.1	83.9	0.0	77.9	22.1	0.0
11	17.1	82.9	0.0	70.5	26.0	3.5
12	19.2	80.8	0.0	65.0	26.0	3.5
13	20.4	79.1	0.5	62.2	37.8	0.0
14	17.1	82.9	0.0	65.4	33.7	0.4
15	14.4	85.6	0.0	63.7	34.6	1.7
16	20.8	78.6	0.6	71.7	28.3	0.0
17	18.7	80.6	0.7	69.5	22.5	7.3
Total	**22.9**	**76.9**	**0.2**	**76.1**	**22.6**	**1.2**

*For list of 17 states, see text.

and in different locations, with the use of different sample designs, logistic arrangements, and examiners.

The first presentation of HHANES dental data showed a close similarity between the distribution of dental caries among Mexican-Americans residing in the Southwest and that of other groups in the region examined by NIDR in 1979-1980. The surface-specific distribution of dental caries in Mexican-American children also mirrored that of children who participated in the National Preventive Dentistry Demonstration Project (*9,10*) and children in the NIDR survey (*22*). In HHANES, the occlusal surfaces of molars were the most susceptible tooth surfaces to decay. The findings from HHANES support the observation of Graves and Stamm (*23*) that dental caries is now a disease predominantly of occlusal surfaces of molars. HHANES results showed that few anterior teeth of Mexican-Americans were affected by caries, and few children's teeth were extracted because of caries—findings similar to those from the NPDDP (*10*).

Because of differing patterns concerning the use of dental services and the observation that Mexican-Americans seek dental services less frequently than other groups surveyed (*14,15*), Mexican-American children in the Southwest were expected to have significantly more decayed teeth than other children residing in the same region. The data from HHANES, however, showed that for Mexican-American children, filled teeth were the main contributor to the total DMFS scores. The ratio of filled surfaces to the total DMFS scores was approximately 66%. In the NIDR survey, this ratio for Region V children was approximately 74%, whereas for Region VII (Washington, Oregon, and California) in the NIDR survey, the ratio was over 90%. These differences may be associated with the lower average income of Mexican-Americans when compared with regional average incomes.

Prevalence of Dental Caries

The predominant feature of dental caries in the Mexican-American children was the overall low level of disease and its clustering in occlusal surfaces of molars. Children (all races) from the southwestern states have long had a lower prevalence of dental caries than children from other parts of the country (*6,7,9,10*), and a comparison of the southwestern portion of HHANES and a statewide survey in South Carolina, also conducted during 1982-1983, shows that these differences still remain (*24*). Although numerous explanations have been promoted for the low prevalence of dental caries in certain geographic regions (*25,26*), none have been scientifically tested within the context of the multifactorial causes of caries. One likely explanation for the lower prevalence of dental caries in the Southwest is the wide availability of naturally fluoridated water supplies (*27*). Since the survey population's residential history was not collected in HHANES, the effect of exposure to fluoridated water cannot be explored further. In HHANES, the reported dietary and nutritional intake of Mexican-Americans has been assessed, but the data collected have not yet been comparatively analyzed.

Despite the overall low level of dental disease in Mexican-American children, only 14.6% of the 17-year-olds were caries-free, and over 50% of those children had five or more decayed or filled teeth. Also, of the estimated 5.6 million decayed, missing, or filled teeth of Mexican-American children, approximately 3.3 million (58.9%) were contributed by about 26.0% of the children. This finding concurs with the results of the NPDDP study (*10*): a relatively small percentage of the children are highly susceptible to dental caries, but the majority are not.

The distribution of dental caries in southwestern Mexican-Americans raises a number of issues on the effectiveness and economics of different preventive programs. Because of the Mexican-Americans' concentration of dental decay on occlusal fissures, the use of fissure sealants as a preventive procedure seems most appropriate. Based on HHANES data, approximately 77.0% of the decayed or filled occlusal surfaces in 17-year-olds had sound proximal surfaces and, therefore, could theoretically have been saved from fissure caries with the use of fissure sealants. These findings strongly support the recommendations of the Council on Dental Research of the American Dental Association concerning sealants (*28*).

Although income was not a significant predictor of total DMFS scores, analysis showed that Mexican-American children from families with low annual income had about two times more decayed teeth than children from high-income families. This high level of unmet dental need among children from low-income families was also observed in the National Preventive Dentistry Demonstration Program (*29*). HHANES data strongly support the need for dental public health programs that give special attention to these children.

Periodontal Disease

Overall, HHANES data showed very little periodontitis (pocketing) but a high percentage of mild gingivitis among Mexican-American children. These results suggest that Mexican-Americans need considerably more education concerning dental health.

SUMMARY

Analysis of Southwestern HHANES data showed that the prevalence of dental caries among Mexican-American children is similar to that of children of the same age examined during the NIDR survey (1979-1980), despite a lower level of restorative treatment. Approximately 50% of Mexican-American children 17 years of age, however, had five or more teeth that were either decayed or filled. Occlusal surfaces of molars were the most susceptible teeth to decay; few anterior teeth were affected. This distribution of dental caries strongly supports the use of fissure sealants on molar teeth. Children from low-income families had two times more decayed teeth than children from high-income families. Mild gingivitis and poor oral hygiene were more prevalent in the Mexican-American children than in the child population for the region examined during NHANES I in 1971-1974. Children from high-income families had better periodontal health than those from low-income families.

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